Pulmonary embolism presenting as adult respiratory distress syndrome—support for a hypothesis

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Summary

Adult respiratory distress syndrome (ARDS), or non-cardiogenic pulmonary oedema, has only rarely been associated with pulmonary embolism. In this case study the association is further documented and the occurrence of pulmonary oedema confined to un-obstructed portions of the pulmonary capillary bed is illustrated.

Introduction

Adult respiratory distress syndrome (ARDS), or non-cardiogenic pulmonary oedema, is known to complicate classic pulmonary embolism (Robin, Cross and Zelis, 1973). It has been well documented in animal experiments (Singer *et al.*, 1957; Swenson, Lamas and Ring, 1965; Parmley, North and Ott, 1962) but is rare in man (Meth *et al.*, 1975). A number of mechanisms have been proposed including the notion that major occlusions of the pulmonary vasculature may result in pulmonary hypertension with transmission of the high pulmonary arterial pressure to unobstructed portions of the pulmonary capillary bed (Robin *et al.*, 1973). Nevertheless, this has not been demonstrated in man and the present case is presented in support of this hypothesis. The patient described here had angiographically documented pulmonary emboli and developed pulmonary oedema confined to unobstructed segments and in the presence of a normal pulmonary capillary 'wedge' pressure.

Case report

A 61-year-old man underwent radical excision of a malignant squamous cell tumour of the oropharynx. His past history was significant for myocardial infarcts sustained in 1962 and 1965, and systemic arterial hypertension under treatment with hydralazine. The pre-operative chest radiograph was normal except for moderate cardiomegaly (cardiac diameter 16 cm) (Williams, 1977). The surgical procedure included a prophylactic tracheostomy with hemimandibulectomy, hemimaxillectomy and radical neck dissection. His postoperative course was unremarkable for the next 72 hr but following transfer from the intensive care unit to the ward, the patient was found to be tachypnoeic and hypotensive (blood pressure 80/60 mmHg) with a heart rate of 150/min. Arterial blood gases breathing room air were abnormal: pH 7·32, P_{O_2} 52 mmHg, P_{CO_2} 28 mmHg. A chest radiograph (Fig. 1) revealed bilateral acinar shadows sparing both lower zones and consistent with pulmonary oedema. There was no increase in heart size. An electrocardiogram showed no change from the preoperative record apart from a sinus tachycardia. A diagnosis of pulmonary oedema was made and 80 mg of frusemide was administered intravenously, with a resulting diuresis of 2 litres over the next 2 hr. Nevertheless no improvement was apparent in the patient's clinical status, arterial blood gases or chest radiograph. A flow-directed pulmonary artery catheter was inserted and the pulmonary artery pressure was found to be 25/5 mmHg, with a mean of 16 mmHg. The pulmonary capillary 'wedge' pressure was 3 mmHg.

Because of the confusing clinical and radiographic picture a pulmonary angiogram was performed (Fig. 2). This demonstrated intraluminal filling defects in arteries to both lower lobes coincident with the non-oedematous areas (Fig. 3). A diagnosis of pulmonary embolism was thus confirmed and anticoagulant therapy with intravenous heparin was instituted. The normal pulmonary cap-
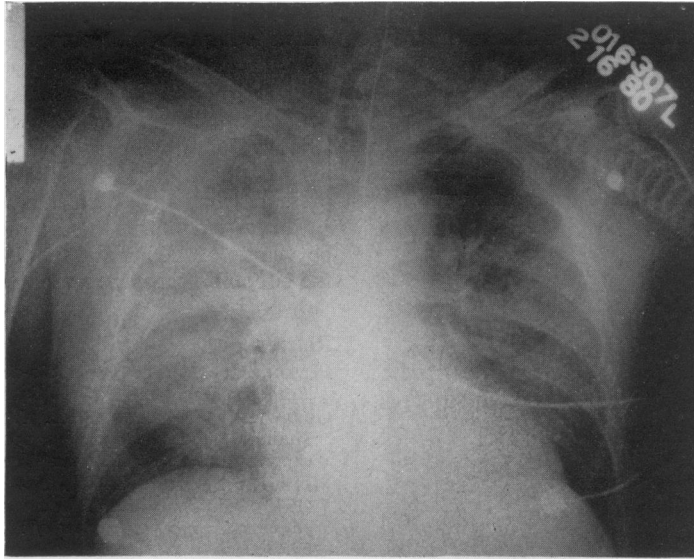
FIG. 1. Anterioposterior chest radiograph showing bilateral diffuse homogeneous opacities consistent with an alveolar filling process such as pulmonary oedema. Both lower zones are noticeably spared.

illary 'wedge' pressure excluded left ventricular failure, and the radiographic abnormality was interpreted as non-cardiogenic pulmonary oedema. The patient was subsequently managed by fluid restriction and artificial ventilation. A fractional inspired

FIG. 2. Pulmonary angiogram showing absent filling of vessels in both lower zones particularly on the right. Intraluminal filling defects (→) were identified in branches of the right and left basal arteries.

O_2 concentration (F_1O_2) of 1·0 was initially required to maintain an arterial Po_2 of 50 mmHg, so that positive end expiratory pressure (PEEP) of 15 cm H_2O was gradually introduced. Over the next 48 hr oxygenation improved (together with the radiographic appearance), and the F_1O_2 was reduced to 0·4 and the level of PEEP to 10 cm H_2O. On this regime arterial blood gases were: Po_2 59 mmHg; Pco_2 35 mmHg; pH 7·44. Five days after respiratory failure had first developed broad-spectrum antibiotic coverage was begun for fever of uncertain source. One day later the patient suffered a cardiac arrest and could not be resuscitated. Permission for a post-mortem examination was denied.

Discussion

Pulmonary oedema is recognized as a complication of pulmonary embolism (Short, 1952; Felson, 1973) but usually occurs in those patients with left ventricular dysfunction (Yuceoglu *et al.*, 1971) and in these individuals is thought to be due to left ventricular failure. Non-cardiogenic pulmonary oedema, or ARDS, occurs in a wide variety of clinical settings such as hypovolaemic shock, major trauma and septicaemia. It has been described as a complication of classic pulmonary thromboembolism by Windebank and Moran (1973), and in more detail in a case report by Meth *et al.* (1975), though in this instance no pulmonary capillary 'wedge' pressures were obtained. In the present case pulmonary embolism was documented by angiography and the normal capillary 'wedge' pressures exclude left ventricular

failure as a cause of the pulmonary oedema. Although it might be argued that therapy with frusemide early in the course of the respiratory failure had 'cured' any left ventricular failure that was present, it is noteworthy that the patient's clinical state did not improve. At no time was the 'wedge' pressure greater than 10 cm H_2O and it therefore seems clear that this was truly a case of non-cardiogenic pulmonary oedema or ARDS.
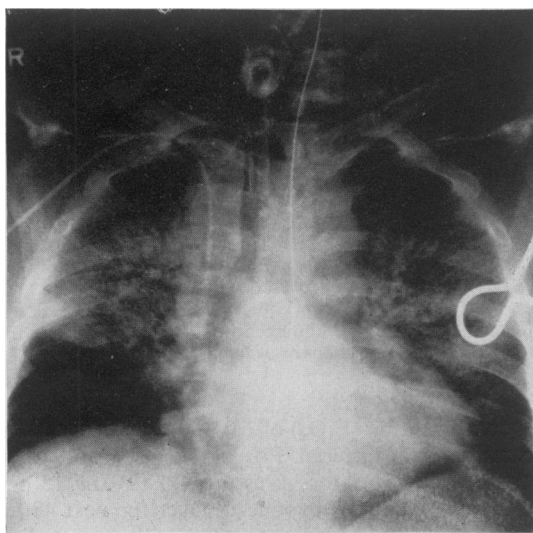

FIG. 3. Anterioposterior chest radiograph taken immediately before catheterization of the pulmonary artery to show the limited distribution of the radiodensity.

The case is interesting not only because this ARDS was associated with pulmonary embolism but also because the alveolar oedema was initially confined to areas not obstructed by clot. This has implications for any theory of pathogenesis. It has been proposed that the release of vasoactive substances from clots may lead directly to increased capillary permeability (Gurewich, Cohen and Thomas, 1968), or to venoconstriction and an increased capillary hydrostatic pressure (Swenson, quoted by Robin, 1970); in addition, it is possible that fibrin microemboli may injure pulmonary capillaries (Saldeen 1976). All these possibilities would, of course, be expected to produce oedema 'downstream' of the obstruction. Another mechanism may be maldistribution of blood flow causing some areas of lung to be overperfused. In this instance, the unobstructed

areas of the pulmonary circulation would be subjected to high regional pressures resulting in interstitial and alveolar oedema (Ohkuda *et al.*, 1978). Because the obstructed areas were unaffected by oedema in the present case, this hypothesis seems tenable. The absence of overall pulmonary hypertension does not exclude the possibility since local pressures may have been transiently quite high.

This case confirms that pulmonary embolism may be a cause of ARDS and that obstructed portions of the circulation may be spared.

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